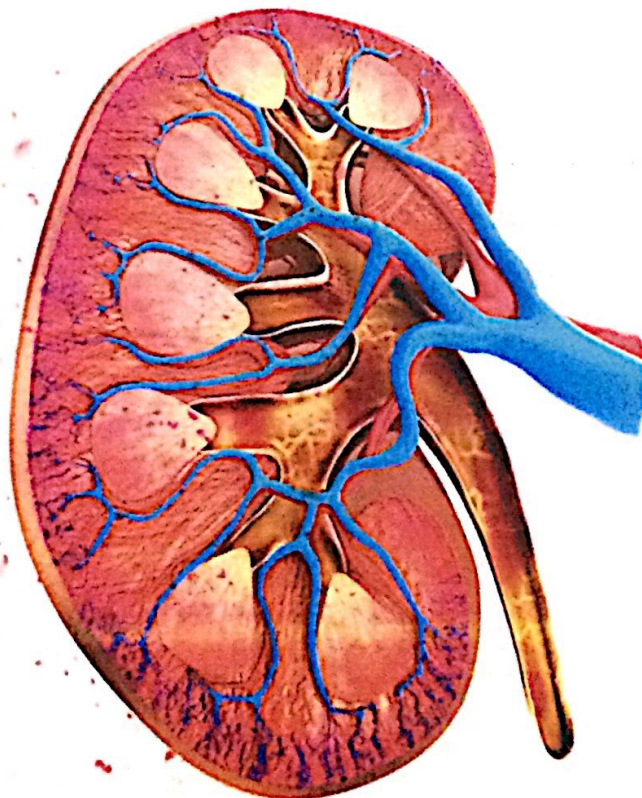


Easy Pathology

— Dr. Abdelrahman Khalifa —

Diseases of the Kidney



Ain Shams 2017 - 2018

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Congenital diseases

Agenesis: Absence of **one** kidney. The other one shows **compensatory hypertrophy**.

Hypoplasia: Failure of maturation. The kidney is **small** (reduced number of nephrons).

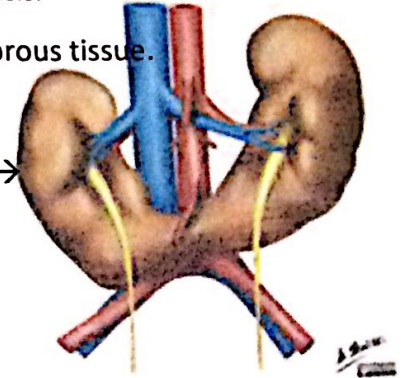
Ectopic kidney: Failure of ascent → **pelvic** → kinked Ureter → Hydronephrosis.

Horse-shoe kidney: Fusion of both kidneys at lower pole by renal or fibrous tissue.

Double Ureter with single or double pelvis

Aberrant renal artery enter at one pole → cross pelviureteric junction → Hydronephrosis & hypertension

Ureteric stricture



Congenital polycystic kidney

Adult polycystic kidney

Infantile polycystic kidney

More common

Less common

A.D. (PKD1 gene) → polycystin-1 (cell-cell & cell matrix adhesion gene)
cystic dilatation of some convoluted tubules

A.R. (PC2 gene) → cystic dilatation of most of the collecting tubules

Gross: Large kidney – Multiple cysts (3-4 cm) – contain clear fluid or blood

Gross: Large kidney – Multiple cysts (small)

Micro: Cysts lined with tubular epithelium (cuboidal /columnar)

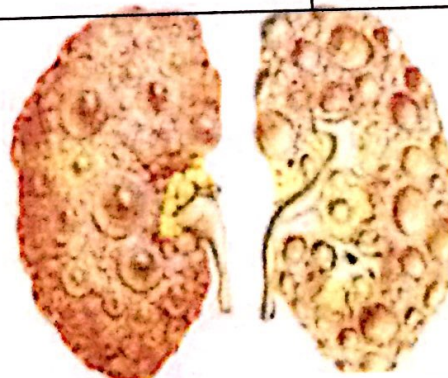
Micro: Cysts lined with tubular epithelium

Complications:

- **Hypertension** (by the age of 30-40) → left sided Heart failure
- **Hematuria**
- **Chronic renal failure**
- **Congenital association with cerebral aneurism 30%**

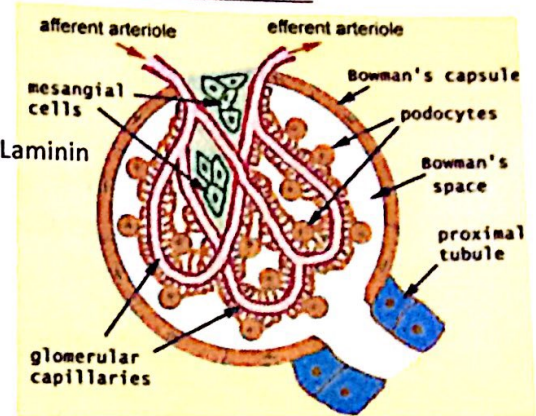
Complications:

- **Acute renal failure** → fatal
- Associated with congenital **hepatic fibrosis** & **Liver cysts**



Structure of glomerulus:

- Fenestrated **endothelium**
- Glomerular **basement membrane** (3 laminae) of Collagen IV, & Laminin
- **Podocytes** with (**foot processes** separated by filtration slits)
- **Mesangial cells**

**Glomerulonephritis**

Structural and functional diseases of glomeruli

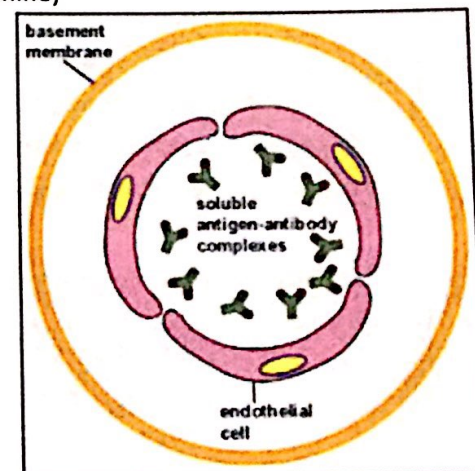
Pathogenesis of GN

1ry (affecting kidneys only)

2ry (SLE, Infections, Tumors & Lymphomas, D.M, gold salt & penicillamine)

- **Circulating immune complex**

- **Ab** against **circulating Ag**, The circulating Ag could be:
 - **Exogenous**: Strept., Syphilis, malaria
 - **Endogenous**: SLE
 - **Unknown**: associated with **HBV**
- e.g. Post streptococcal GN



- **In situ immune complex**

- Immune complex is **formed in glomeruli**, not in circulation
- Ab binds with **fixed Ag** in glomeruli
- Fixed Ag could be
 - **Endogenous**: Antineutrophilic cytoplasmic antibody (ANCA) complex in SLE
 - **Exogenous**: Endostreptosin of A Streptococcus

Both Circulating & in situ immune complex → **deposit in glomeruli**:

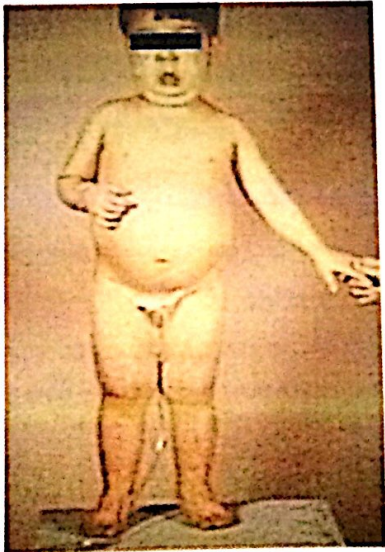
- **Mesangial deposit**
- **Sub-endothelial deposit**
- **Sub-epithelial deposit**
- Immune complex is revealed with **E/M** → **granular deposit** → activates complement → Glomerular injury

- **Anti GBM antibodies:**

- e.g. **Good Pasture** syndrome
 - Ab against basement membrane (**Anti GBM Ab**)
 - Damage of capillary basement membrane → **Hematuria**
 - Ab against pulmonary capillaries → **haemoptysis**
- **Liner** deposit by E/M

The results of immune mediated Glomerular injury

- Leukocytes recruitment (**inflammation**) → mediators & cytokines → proliferation of cells & capillary injury → **Hematuria (Nephritic)**
- **Podocyte injury** → Effacement (fusion of foot processes) → **Proteinuria (Nephrotic)**

	Nephritic	Nephrotic
Mechanism	-Leukocytes recruitment (inflammation) → mediators & cytokines → <u>proliferation of cells & capillary injury</u> → Hematuria -Reduction of GFR → Oligurea & Azotemia -Ischemic kidney → Release of Renin → Hypertension	-Podocyte injury → Effacement (fusion of foot processes) → Proteinurea -HypoAlbuminemia → generalized edema -Drop in plasma volume → Release of Renin → <u>Water retention</u> in a background of low protein → more edema -Liver secretes Lipoprotein → Hyperlipidemia
Clinical Picture	• Hematuria (smoked urine) • Mild proteinurea (not severe because of oligurea) less tha 1 gm • Mild hypoproteinemia • Mild edema (orbital) • Oligurea • Hypertension • Azotemia (mild transient uremia) 	• severe proteinurea >3.5 g/day • hypoproteinemia < 3gm/dl • generalized edema • Lowered immunity (<i>loss of Immunoglobulin</i>) • Hyper-lipidemia (<i>Lipoprotein secretion</i>) 

Gross	Large – Pale kidneys (edema) Petechial cortical spots (hemorrhage)	Large – Pale kidneys (edema) yellow spots (Lipid)
Micro & E/M	-Capillaries: Dilatation & injury -Glomeruli: Inflammatory cells & RBCs -Proliferated cells (endothelial – Mesangial) -Tubules: contain Casts -Immune complex deposit	-Podocytes: Effacement (loss of foot processes) -Tubular epithelium: Hyaline droplets (absorbed protein) Fat vacuoles (absorbed lipid) -Tubular lumen: Protein cast -Interstitial tissue: edema
Causes	• Acute diffuse prolif. GN • Rapidly progressive GN (Crescentic) • IgA nephropathy (Berger's) 2ry nephritic: • SLE • Henoch Schoenlein purpura	• Minimal change GN • Membranous GN • Membrano-prolif. GN • Focal segmental GS • IgA nephropathy 2ry nephrotic: SAD • SLE • Amyloidosis • DM

Acute diffuse proliferative GN

(Acute post-streptococcal)

Aetiology: B-hemolytic strept., nephrogenic strains (following URTI), in children

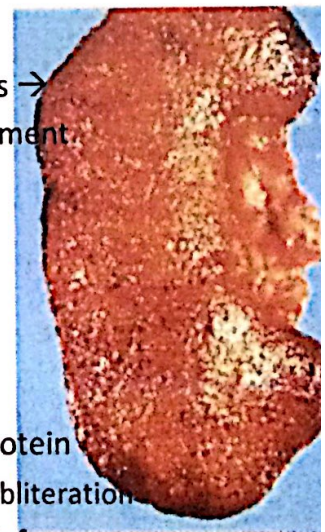
Pathogenesis:

Group A Beta **Streptococcal** infection (sometimes staph) → after 1-2 weeks → Antibodies → immune **complex** → deposit in glomeruli → activate complement pathways → inflammatory reaction & glomerular injury

Gross: both kidneys enlarged – pale – red spots

Microscopic: Affecting all glomeruli

- **Capillaries**: dilated
- **Bowman's capsule**: contain RBCs & neutrophils, monocytes, few protein
- **Proliferation**: swelling of Mes. cells & endothelial cells → capillary obliteration
- **Tubules**: contain casts (red cell casts) → the casts are common with fever



E/M: immune complex (**humps & lumps**) – **subepithelial** (under podocytes)

I/F: **Granular** deposits **IgG** & complement

C/P: nephritic (describe) + elevated ASO + low complement

Fate:

- **Resolution** within 14 days in 95% of children, and 60% of adults
- **Rapidly progressive GN** → acute renal failure and death
- Chronic G.N.

Rapidly progressive GN

(Crescentic GN)

Nephritic syndrome, with rapid deterioration of renal function & formation of Crescent

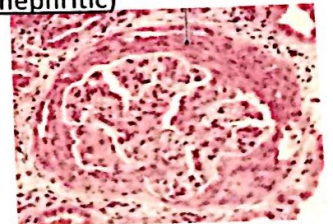
Aetiology:

- **Type I** : Anti GBM Good pasture (Linear deposit of IgG & C3)
- **Type II**: Immune complex (Following post-strept GN, or other causes of nephritic)
- **Type III**: Pauci immune : Vasculitis : PAN or Wagner (No Ab or complex)

Gross: the same

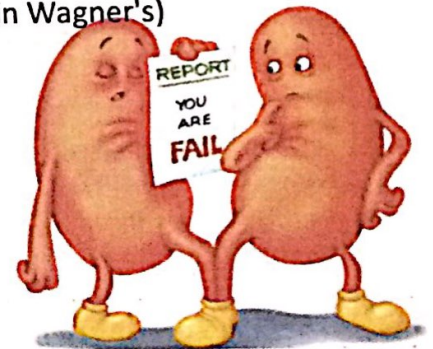
Microscopic:

- Similar to nephritic (describe)
- **Crescent formation** : proliferation of **parietal** cells in bowman's space + fibrosis → Sclerosis in advanced cases → tubular atrophy
- **Blood vessels** : changes of **malignant hypertension**
- **Segmental necrosis of glomeruli** - **fibrinoid** necrosis in B.V. (in Wagner's)



E/M & IF : Linear deposits in **type I** - **Granular** deposits in **type II**

Fate: Rapidly progressive in few months → acute renal failure (fatal)



IgA Nephropathy

The most common glomerular disease

Aetiology: (following non specific **URTI**) , in children or Adults (10-29 years)

Pathogenesis: **Abnormal IgA** (1ry or 2ry hepatic) → deposit in **mesangium** →

Complement activation

Gross: The same..

Microscopic: Variable microscopic findings

- **Normal** glomeruli in some cases
- **Mesangial** proliferation (mesangioproliferative)
- **Focal proliferative** GN (describe)
- **Crescentic** (in advanced cases)



E/M: Mesangial deposits

I/F: Ig A, few amounts of IgG or Ig M

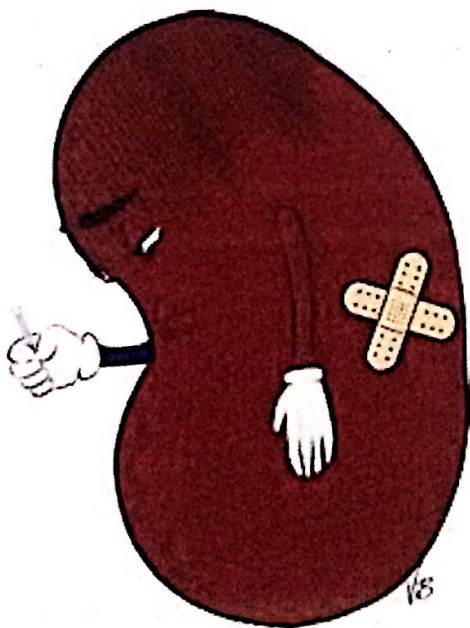
C/P: nephritic (Hematuria for days) → recurrent every few months

Prognosis:

Good - **Bad** in case of (**crescentric** – Old age – Heavy proteinuria & hypertension)

Clinical presentations of renal diseases:

- **Nephritic** (describe)
- **Nephrotic** (describe)
- **Acute R.F.** (Hypertension – Oliguria or anuria) → **reversible**
- **Chronic renal disease and chronic R.F.** (Anemia – Uremia – Osteoporosis)
- **Tubular defect** (Polyuria & Nocturia – electrolyte imbalance)
- **UTI** (Fever – Loin pain – Dysuria – Pyuria)
- **Stone & obstruction** (Colic – Hematuria – Acute renal failure)
- **Asymptomatic** (Hematuria – proteinuria – pyuria)



Nephrotic

	Minimal change disease (Lipoid nephropathy)	Membranous GN	Membr-prolif. GN		Focal segmental GS
Incidence	More in Children	More in Adults	Adults & children		More in Adults
Aetiology & Mechanism	Unkown may be T-cell mediated	1ry: idiopathic 2ry to: DM – HBV- Bilharziasis – Aspirin – Cancers (instu immune complex)	Type I: HBV, SLE (immune complex) Type II: Abnormal complement		1ry: idiopathic 2ry to: AIDS -Heroin – IgA nephropathy
Micro	Nephrotic (describe?)	Nephrotic (?) + Thick B.M. and spiky (hair-comb)	Nephrotic (?) Thick BM & splitting (tram-track) + Proliferated cells (Mesangial& endothelial)		nephrotic (?) + Some glomeruli Some capillaries Show hyalinosis & sclerosis (irreversible)
E/M	Fusion of foot processes of podocytes (effacement)	Fusion of foot processes Sub-epithelial granular deposits	Type I Sub endoth.	Type II Intra-membrane Deposit (Dense Deposit Dis)	Fusion of foot processes
I/F	No Ig	IgG & complement	IgG, C3	Granular C3	IgM & C3 in sclerotic segment
C/P	Nephrotic (describe) selective Allbuminurea	Nephrotic (describe)	Nephrotic (describe) Or Nephritic (describe)		Nephrotic (describe) + nephritic in some cases
Fate	Good in Children -Respond to cortisone -Chronic RF not common	-Respond to cortisone -Chronic RF (40%)	Type I : 40% R.F. Type II : worse		Bad prognosis chronic RF

Chronic GN & Chronic kidney disease

Aetiology: End stage renal disease following different types of GN (except RPGN)

Gross: Size: Symmetrically reduced (contracted) Surface: granular & cystic

Microscopic:

- **Small & medium Arteries:** Thick wall (due to hypertension)
- **Glomeruli:** atrophy, fibrosis & sclerosis
- **Interstitial tissue:** chronic inflam. Cells + fibrosis
- **Tubules:** atrophy

C/P:

- **Azotemia or Uremia** (elevated urea & creat)
- **Hypertension** (?)
- Picture of nephrotic or nephritic

Fate: Chronic renal failure → fatal



Tubulo-interstitial nephritis:

- Pyelonephritis
- TB nephritis
- Drug induced nephritis
- Acute tubular injury

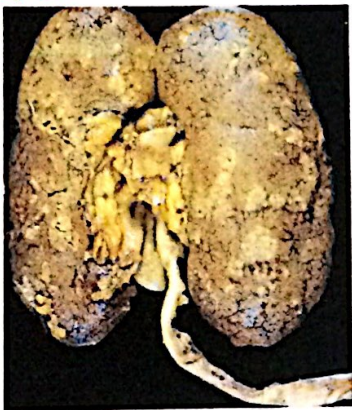
Drug induced interstitial nephritis

Antibiotics & analgesics

Acute Interstitial nephritis	Chronic Analgesic nephropathy
Synthetic antibiotics (synthetic Penicillin) Others: Diuretics, Cimetidine	Aspirin + Acetaminophen for long duration
-Drug hapten + binds with tubular protein → Ag - IgE mediated or Type IV hypersensitivity	- Acetaminophen metabolite → binding & oxidative damage - Aspirin → Anti Pg → papillary vasospasm → necrosis
Granulomatous reaction 	Gross: Papillary necrosis (yellow brown & friable) Micro: Coagulative necrosis fate → Chronic R.F. & hypertension 

Pyelonephritis

Suppurative inflammation of the **pelvicalyceal system** and parenchyma

	Acute pyelonephritis	Chronic pyelonephritis
Incidence	More common in females (short & wide urethra)	
Aetiology & PF	Organism: KEEPS (Klebsiella, E.coli , Enterobacter, Proteus, Srept fecalis), Aerobacter Route: <u>Ascending</u> infection - Blood P.F. : Vesico-ureteric reflux (congenital- acquired) - catheterization – obstruction & stones DM & low immunity - Pregnancy (compression) - Bilharziasis Chronic Pyelonephritis : Obstructive or Reflux-induced	
Gross	Size: large Pelvis & calyces: Inflamed (pus) Medulla: Yellow streaks Cortex: yellow spots 	Patchy fibrosis (scarring) Pelvis & calyces : Scarring → marked calyceal deformity & papillary blunting 
Micro	Interstitial: Inflam. cells (pus) Tubules: pus Arterioles: dilated Glomeruli: normal Calyces: Acute Inflammation	Interstitial: Inflam cells (plasma , macrophage, pus) Tubules: dilated contain cast (thyroidisation) PAS +ve Arterioles: Sclerosis Glomeruli: Sclerosis Calyces: chronic Inflammation & fibrosis
C/P & complications	Fever – Loin pain - Dysurea – Pyurea with Blood & protein casts Fate: -Resolution -Acute RF -Chronic pyelonephritis - Pyonephrosis (with obstruction)	- Pyurea , & protein casts -Chronic RF & hypertension Pyelography → damaged calyces

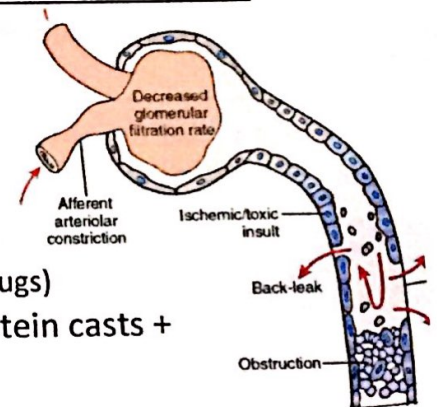
TB of the Kidney

- **Aetiology:** 2ry TB (blood spread – ascending genitourinary)
- **Gross:**
 - **TB Pyelonephritis:**
 - Medullary Tubercles in papillae → caseation
 - Caseation of medulla → open into renal pelvis → **TB cavitation** in medulla
 - In case of U.T. obstruction → **TB pyonephrosis** (dilated pelvis & calyces)
 - **Miliary:** multiple small uniform nodules
- **Micro:** Caseating granuloma (*describe?*) - in Miliary (non caseating)
- **Clinical:** Sterile pyuria, Hematuria & proteinuria



Acute Tubular injury (ATI)

- Most **common** cause of **acute renal failure**
- **Anoxic** (Shock – Burn – ABO mismatch – Injury)
- **Toxic** (**Mercury** chloride – **CCL4** – Phosphorus – some drugs)
- Necrosis of tubular epithelium (proximal) + protein casts + interstitial edema



Diabetic Nephropathy

- **Pyelonephritis**
- **Papillary** necrosis
- **Arteriosclerosis**
- **Glomerulosclerosis** '**Nephrotic**' (in long standing D.M. >12 years)
 - **Diffuse Glomerulosclerosis**: Thick membrane + Mesangial proliferation
 - **Nodular** (**Kimmelsteil-Wilson**): Mesangial **deposits** proteins & Lipid



Renal Amyloidosis:

Deposition of insoluble Amyloid fibrils (1ry or 2ry) → Mainly **Nephrotic**

Lupus Nephritis

Most **common** complication of SLE

Pathogenesis: Immune complex (Anti-DNA Ab) → Proliferative lesion & vascular necrosis (**Nephritic**), or **Nephrotic** lesions

C/P : Nephritic, or nephrotic, or Chronic R.F.

E/M & I/F : Granular deposit Subendothelial – Mesangial (IgG)

Mesangial deposits → **Wireloop Glomerulosclerosis**

Classes:

I: Minimal Mesangial

II: Mesangioproliferative

III: **Focal** proliferative Lupus

IV: Diffuse proliferative Lupus > 50%

V: membranous

VI: Sclerosing lupus

Diffuse proliferative Lupus (hypercellular glomeruli 'describe' + **wire loop**) → may lead to **crescentic** 'describe' (fatal)



Hypertension

	Benign nephrosclerosis	Malignant Nephrosclerosis
Gross	Size: Reduced (bilateral) Surface: <u>granular</u> - small cysts C/S: Atrophic cortex No demarcation between cortex & medulla	Size: Normal Surface: Smooth C/S: red spots (flea-bitten)
Micro	<u>Arteries:</u> Hyperplasia (fibro-elastic) <u>Arterioles:</u> (Hyaline arteriosclerosis) → thick wall, narrow lumen <u>Nephron:</u> atrophy , some nephrons → compensatory dilatation	<u>Arterioles:</u> Hyperplasia (onion skin) – Fibrinoid necrosis (necrotizing) <u>Nephron:</u> necrotizing glomerulitis
C/P	Oligurea – diluted urine – proteinurea	Cause of death 90% R.F. (uremia) Systemic complications of hypertension (Heart failure, visual impairment, encephalopathy)

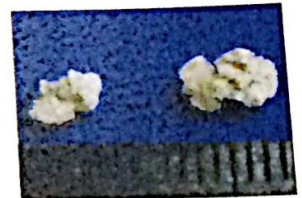
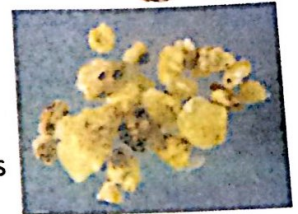
Renal artery stenosis:

Atherosclerosis – Vasculitis – Radiation – Fibromuscular dysplasia
 → renal ischemia → **2ry hypertension**

Urinary calculi – stones (urolithiasis)

Aetiology & pathogenesis ASI

- **Abnormal composition of urine**
 - Dehydration
 - Increased salts (hypercalcemia – oxalate – hyperurecemia in gout – congenital cystinurea)
 - Decreased **solubility factors** (e.g. pyrophosphate, nephrocalcin)
- **Stasis** due to obstruction → infection
- **Infection**
 - Shedding of epithelium (*Predisposed by vit A deficiency*) → Nidus
 - PH changes
 - Bacilli → Acidic urine → deposit oxalate & urate stones
 - Cocci → Alkaline urine → deposit CaPh stones



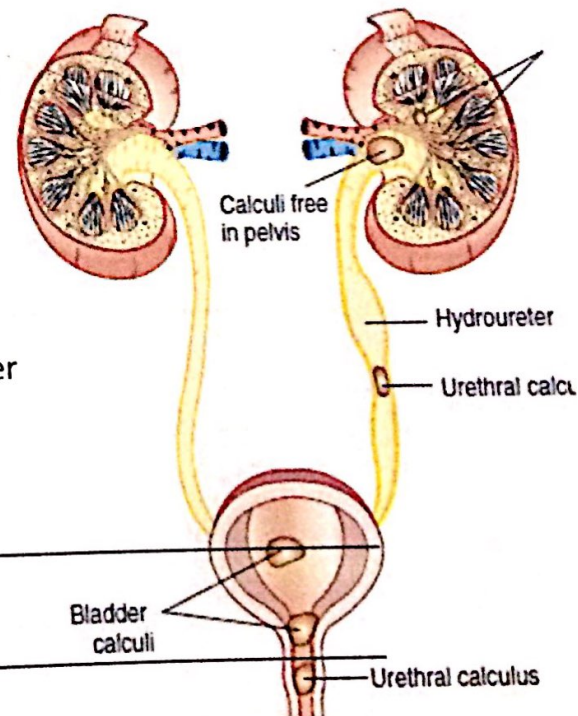
Types of stones

- **Oxalate** stone : Hard, spiky & brown
- **Urate** stone 7%: Hard – rounded small or staghorn– radiotranslucent – yellow
- **Ca Phosphate**: Large - white friable chalky (with infection & alkaline urine)
- **Struvite** is a mixed stone **CAMP** (calcium, magnesium, Ammonium, phosphate) 10%
- **Cystine** 1% : small yellow

The most common types : Ca phosphate & Ca oxalate (80%)

Complications

- Loin pain
- Renal colic
- Hematuria (commonly oxalate stone)
- Obstruction → Hydronephrosis & hydroureter
- Calculus anuria
- Chronic Cystitis
- Squamous metaplasia → carcinoma

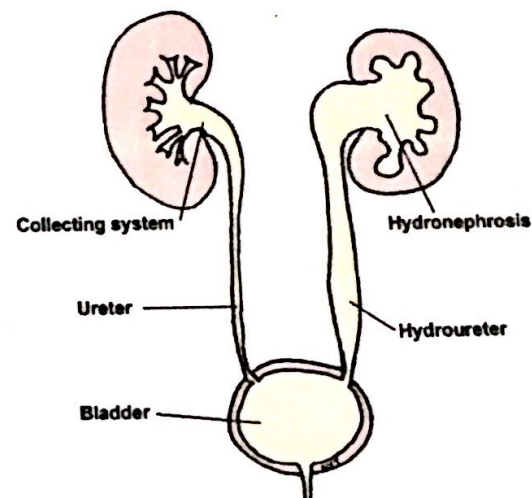


Hydronephrosis

Dilatation of **pelvi-calyceal** system due to **partial or intermittent** urinary tract obstruction

Causes of urinary obstruction:

- Renal : stag-horn stone, tumors
- Ureteric
 - **Congenital** (e.g. stricture – abarrent renal artery-ectopic kidney)
 - **Inflammation & fibrosis** (e.g. Bilharziasis, TB, gonorrhea)
 - **Stone**
 - **Tumors & endometriosis**
 - Compression by pregnant uterus, or cancer cervix or retroperitoneal fibrosis
- Bladder
 - **Congenital** (e.g. diverticula)
 - **Inflammation & fibrosis** (e.g. Bilharziasis, TB)
 - **Stone**
 - **Tumors**
- Urethral
 - **Congenital**
 - **Inflammation & fibrosis** (e.g. Bilharziasis)
 - **Stone**
 - **Tumors**
 - **Prostatic enlargement** (e.g. hyperplasia or tumors)



Morphology

- Kidney: **enlarged** – **Atrophic cortex**
- Pelvis and calyces: **dilatation**– **Clubbing** of calyces

Pyonephrosis

Dilated pelvi-calyceal system due to obstruction & **pyogenic** infection

Causes

- Causes of Hydronephrosis → followed by Pyogenic infection
- Acute pyelonephritis → followed by obstruction

Morphology: similar to Hydronephrosis + containing pus

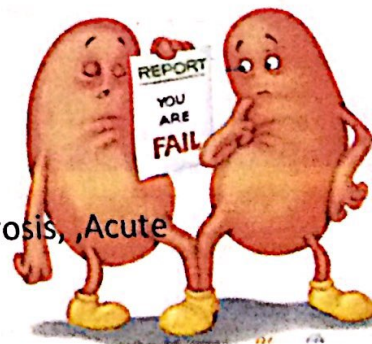
Renal failure

Acute renal failure **Oligurea** <400 ml/day and **Azotemia**

Pre-renal causes (Circulatory failure)

Renal causes (infantile polycystic kidney, RPGN, Acute tubular necrosis, Acute pyelonephritis, papillary necrosis)

Post-renal causes (sudden Complete bilateral obstruction)

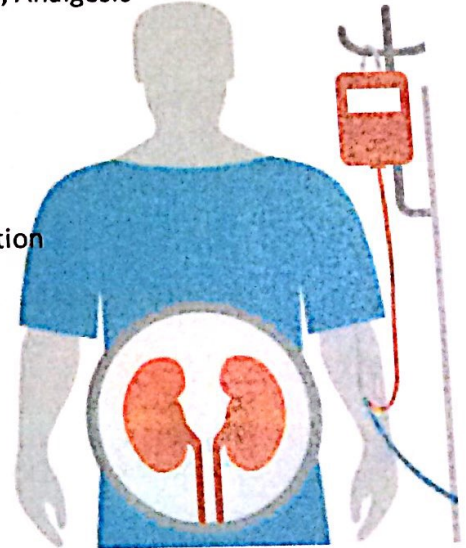


Chronic renal failure

- Adult polycystic kidney
- Chronic GN
- Chronic Pyelonephritis (the most common cause in Egypt)
- Benign hypertension
- Other systemic causes (Diabetic nephropathy ,lupus, Analgesic nephropathy)

Pathological changes in renal failure

- Urea **Encephalopathy** → renal coma , & neuritis
- **Anemia**
- Earthy color of skin (**café au lait**) due to urochrome deposition
- Ammoniacus odor
- Serofibrinous inflammation (pleurisy, pericarditis)
- Uremic pneumonitis
- **Hypertension**
- Heart failure
- GIT inflammation & ulcers
- Renal osteodystrophy
- Lab analysis: **elevated urea , creat**, electrolyte & acid-base imbalance



Renal tumors

Benign :adenoma, fibroma, angiomyolipoma, oncocytoma

Malignant

- Renal cell carcinoma RCC
- Nephroblastoma (Wilm's tumor)
- TCC of renal pelvis
- 2ry metastatic (rare)

Oncocytoma : Large brown mass up to 13cm, contain **central stellate scar**. The cells are derived from PCT , large and acidophilic cells (**oncocytes**)

Renal Cortical adenoma: **small** mass 0.5 cm.

formed of glands with **pap. Projections**. Larger tumors **>3cm are considered malignant.**

Angiomyolipoma : formed of vascular spaces, fat cells, & smooth muscles



Renal cell carcinoma (RCC)

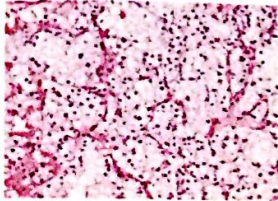
Incidence: more common in **males** > 60y

P.F.: Genetic factors – **Smoking** – Cadmium

P.L.: Polycystic kidney (high risk)

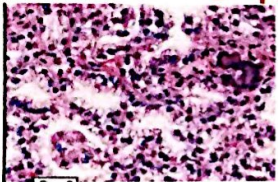
Types:

- **Clear cell RCC (80%)**



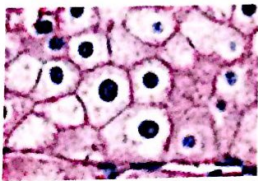
- Genetic mutations (**VHL** gene) : Hemangioblastoma + bilateral multiple RCC 40%
- Originating from **PCT**
- Gross: polar, rounded, golden yellow, with areas of hemorrhage
- Micro: sheets of **clear cells** (glycogen & fat), separated by thin stroma. Nuclei are rounded and dark. Areas of necrosis & hge.

- **Papillary RCC (10%)**



- **MET** oncogene (GF receptor)
- Micro: branched **papillae** covered by malignant cells. The core is fibrovascular may show **Psammoma bodies**.

- **Chromophobe RCC (5%)**



- Multiple chromosomal loss, originating from collecting ducts
- Micro: sheets of **large acidophilic granular cells**, cytoplasm show **halo** around nucleus. Prominent cell membrane.
-

C/P & complications of RCC:

- **Palpable mass**
- **Painless Hematuria** (no dysuria)
- **Pain in Loin**
- **Paraneoplastic syndrome:**
 - Hypertension (Renin)
 - Polycythemia & leukomoid reaction (Erythropoietin stimulating prtn)
 - Cushing (cortisone), hypercalcemia (parathyroid hormone)
 - Feminization or masculinization (sex hormones)
- **Spread:**
 - Direct (left renal vein invasion → **left varicocele**)
 - Lymphatic → para aortic LN
 - Blood → Lung (cannon ball metastasis), B, L, B



Nephroblastoma

(Wilms)

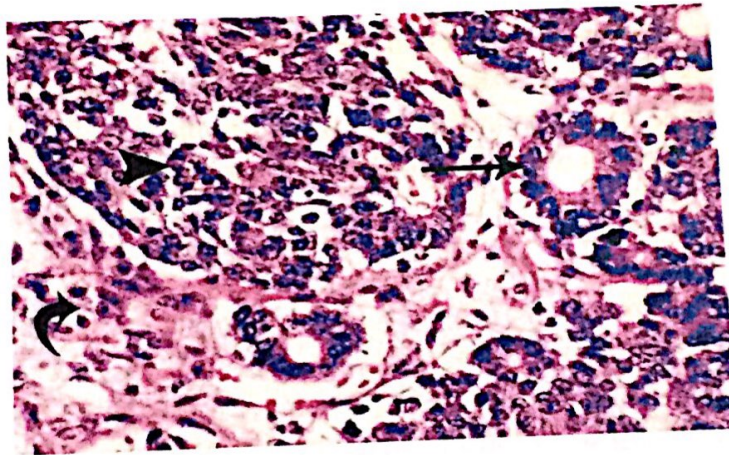
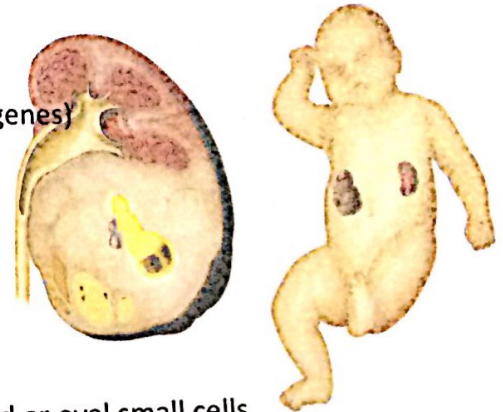
Incidence: children < 6 y (genetic mutations in **WT1**, **WT2** genes)

Origin: remnants of **embryonic** blastemal cells

Gross: Large, greyish, with necrosis & hemorrhage

Micro: Tri-phasic

- Blastemal **embryonic** cells: highly anaplastic rounded or oval small cells
- **Epithelial** component: abortive glomeruloid & abortive tubular structures
- **Mesenchymal**: fibrous, muscle, cartilage, bone



C/P & Complications:

Palpable large mass (intestinal obstruction) – Pain in loin – Painless Hematuria – Spread (D – L – B)

1) A 53-year-old man has passed darker urine for the past week. On physical examination there are no abnormal findings. A urinalysis shows pH 5.5, specific gravity 1.013, 2+ blood, no protein, and no glucose. A urine cytology is performed and there are atypical cells seen. A cystoscopy is performed, but no mucosal lesions are noted. He has a 60 pack year history of smoking cigarettes. Which of the following is the most likely diagnosis?

- A. Adenocarcinoma of prostate
- B. Urothelial carcinoma of renal pelvis
- C. Acute interstitial nephritis
- D. Nodular glomerulosclerosis
- E. Squamous cell carcinoma of penis

2) A 52-year-old previously healthy man has experienced episodes of discomfort with urination for 3 months. There are no remarkable findings on physical examination. Laboratory studies include a urinalysis that reveals 1+ blood. Microscopic urine examination shows numerous RBCs, a few WBCs, and no casts. A urine culture is negative. A plain film radiograph of the pelvis shows a rounded, 1 cm radiopaque lesion in the region of the bladder. Which of the following laboratory test findings is most likely to be present in this man?

- A. Albuminuria
- B. Hypercalciuria
- C. Transaminasemia
- D. Hemoglobinuria
- E. Hyperuricemia

3) A 72-year-old man has been feeling tired and lethargic for 5 months. He has noted increasing hesitancy with urination. On physical examination his prostate is diffusely enlarged. Laboratory studies show sodium 139 mmol/L, potassium 4.0 mmol/L, chloride 104 mmol/L, CO₂ 25 mmol/L, creatinine 1.9 mg/dL, and glucose 81 mg/dL. Which of the following renal abnormalities is most likely to be present in this man?

- A. Cortical atrophy
- B. Glomerulonephritis
- C. Papillary necrosis
- D. Polycystic change
- E. Renal cell carcinoma

4) A 36-year-old woman has urinary frequency with dysuria for the past 4 days. On physical examination she has no flank pain or tenderness. A urinalysis reveals sp. gr. 1.014, pH 7.5, no glucose, no protein, no blood, nitrite positive, and many WBC's. She has a serum creatinine of 0.9 mg/dL. Which of the following is the most likely diagnosis?

- A. Lupus nephritis
- B. Urinary lithiasis
- C. Acute cystitis
- D. Malakoplakia
- E. Urothelial carcinoma

5) A clinical study is performed with pediatric subjects who had a diagnosis of minimal change disease. These patients were observed to have prominent periorbital edema at diagnosis. Laboratory test findings from serum and urine tests were analyzed. Which of the following urinalysis test findings is most likely to have been consistently present in these subjects?

- A. Nitrite positive
- B. Proteinuria >3.5 gm/24 hours
- C. Hematuria with >10 RBC/hpf
- D. Calcium oxalate crystals
- E. Renal tubular epithelial cells and casts

6) A 3-year-old child has become more irritable over the past two months and does not want to eat much at meals. On physical examination the pediatrician notes an enlarged abdomen and can palpate a mass on the right. An abdominal CT scan reveals a 10 cm solid mass involving the right kidney. The resected mass has a microscopic appearance with sheets of small blue cells along with primitive tubular structures. The child receives chemotherapy and radiation therapy, and there is no recurrence. Which of the following neoplasms is this child most likely to have had?

- A. Angiomyolipoma
- B. Renal cell carcinoma
- C. Urothelial carcinoma
- D. Wilms tumor
- E. Medullary fibroma

7) A 5-year-old boy is noted to have increased puffiness around his eyes for the past week, and he has been less active than normal. On physical examination he has periorbital edema. Vital signs include T 37°C, P 75/minute, RR 22/minute, and BP 140/90 mm Hg. A urinalysis reveals sp. gr. 1.010, pH 6.5, no glucose, 4+ protein, no blood, no casts, and no ketones. Microscopic urinalysis reveals oval fat bodies, but no WBC's or RBC's. He improves following a course of corticosteroid therapy. Which of the following renal lesions is most likely to have been present in this boy?

- A. Glomerular crescent formation
- B. Podocyte foot process effacement
- C. Patchy acute tubular necrosis
- D. Hyperplastic arteriosclerosis
- E. Mesangial immune complex deposition

8) A 56-year-old man complains of dull flank pain for the past month. On physical examination he has tenderness to percussion at the right costovertebral angle. Laboratory studies show microscopic hematuria but no proteinuria or glucosuria. A urine cytology shows no atypical cells. A CBC shows WBC count 7800/microliter, Hgb 21.1 g/dL, Hct 63.5%, MCV 94 fL, and platelet count 195,000/microliter. His serum urea nitrogen is 15 mg/dL and creatinine 1 mg/dL. Which of the following radiographic findings is most likely to be present in this man?

- A. Hydronephrosis on intravenous pyelogram

- B. Renal mass on abdominal CT scan
- C. Radiopaque ureteral calculus on an abdominal plain film
- D. Enlarged, multicystic kidneys on abdominal ultrasound
- E. Pelvic abscess below the bladder on MR imaging

9) A 20-year-old previously healthy man has been feeling tired for the past 5 days. He then passes dark-colored urine. On physical examination his blood pressure is 160/90 mm Hg. Laboratory studies show his serum creatinine is 4.4 mg/dL and BUN 40 mg/dL. A urinalysis reveals pH 6, specific gravity 1.011, 3+ blood, 1+ protein, no glucose, and no ketones. On urine microscopic examination there are numerous RBC casts. Which of the following pathologic findings on renal biopsy is most likely to be present in this man?

- A. Glomerular crescents
- B. Widened proximal tubules
- C. Neutrophilic infiltrates
- D. Basement membrane thickening
- E. IgA deposited in glomerular capillaries

10) A 43-year-old man has had increasing malaise for the past 3 weeks. On physical examination he has a blood pressure of 150/95 mm Hg and 1+ pitting edema of the lower extremities to the knees. Dipstick urinalysis shows no glucose, blood, ketones, nitrite, or urobilinogen, and the microscopic urinalysis reveals no RBC/hpf and only 1 WBC/hpf. Additional laboratory studies show a 24 hour urine protein of 4.1 gm. His hepatitis B surface antigen is positive. Which of the following is the most likely diagnosis?

- A. Membranous nephropathy
- B. Systemic lupus erythematosus
- C. Acute tubular necrosis
- D. Diabetic nephropathy
- E. Post-streptococcal glomerulonephritis

11) A 60-year-old woman is admitted with sudden onset of chest pain and is diagnosed with an acute myocardial infarction. There is difficulty maintaining adequate blood pressure and tissue perfusion for 3 days. Her serum lactate becomes elevated. Her serum urea nitrogen increases to 44 mg/dL and creatinine to 2.2 mg/dL. Granular and hyaline casts are present on microscopic urinalysis. Which of the following renal lesions is most likely to be present in this situation?

- A. Chronic pyelonephritis
- B. Acute tubular necrosis
- C. Nodular glomerulosclerosis
- D. Renal vein thrombosis
- E. Minimal change disease

12) A 53-year-old woman has had chronic arthritis pain for the past 3 years. She has taken 2 gm of phenacetin and acetaminophen a day for her pain over that time. She now has increasing fatigue. There are no abnormal findings on physical examination. Laboratory studies show her serum urea

nitrogen is 52 mg/dL and creatinine 5.4 mg/dL. Which of the following pathologic findings is most likely to occur in her kidneys?

- A. Papillary necrosis
- B. Focal segmental glomerulosclerosis
- C. Nephrocalcinosis
- D. Acute interstitial nephritis
- E. Arteriolosclerosis

13) A 39-year-old previously healthy man has the sudden onset of severe right flank pain that comes in waves all night long. When he is seen in the emergency room, after waiting for two hours, he is exhausted. On physical examination there are no abnormal findings. Urinalysis reveals no ketones, glucose, protein, nitrite, or urobilinogen, but blood is present. Urine microscopic examination shows many RBCs but few WBCs. The specific gravity is 1.015 and the pH is 5.5. Which of the following is the most likely diagnosis?

- A. Nodular prostatic hyperplasia
- B. Membranous nephropathy
- C. Ureteral calculus
- D. Renal angiomyolipoma
- E. Urothelial carcinoma of bladder

14) A 15-year-old girl has had increasing lethargy following a bout of the "flu" 3 weeks ago. On physical examination there are no abnormal findings. Her condition does not improve after 3 weeks on corticosteroid therapy, so a renal

biopsy is performed. Microscopic examination shows segmental sclerosis of 3 of 10 glomeruli identified in the biopsy specimen. Immunofluorescence studies and electron microscopy do not show immune deposits. What is the most appropriate advice to give the girl's parents regarding her condition?

- A. She may require a renal transplant in 10 years
- B. She will probably improve with additional corticosteroid therapy
- C. She will likely develop a restrictive lung disease
- D. She has an underlying malignancy
- E. She will improve if she loses weight

15) A 59-year-old man notes blood in his urine for the past week. On physical examination there are no abnormal findings. A urinalysis confirms the presence of blood, but no proteinuria or glucosuria. A urine culture is negative. A cystoscopy is performed, and a 3 cm exophytic mass is seen in the dome of the bladder. A biopsy of this mass is performed and microscopic examination reveals fibrovascular cores covered by a thick layer of transitional cells. Which of the following risk factors is most likely to have led to development of this lesion?

- A. Diabetes mellitus
- B. Recurrent urinary tract infection
- C. Therapy with methicillin
- D. Cigarette smoking
- E. Tuberous sclerosis

16) Glomerulonephritis characterized by proliferation of parietal epithelial cells in most of glomeruli is usually associated with which of the following features:

- A. Proteinuria 5 gm/24 hours , hematuria , oliguria and azotaemia .
- B. Proteinuria 2 gm/24 hours ,hematuria ,anuria and azotaemia .
- C. Proteinuria 6 gm/24 hours,azotaemia and hypertension.
- D. Proteinuria 1 gm/24 hours and hypertension without azotaemia.
- E. Proteinuria 3.5 gm/24 hours with azotaemia and without hypertension

17) A 49-year-old woman has been hospitalized for the past 10 days for treatment of bronchopneumonia. She has developed chills and fever over the

past 2 days. On physical examination her temperature is 38.8°C and she has a diffuse erythematous skin rash. Laboratory studies show serum creatinine 2.2 mg/dL and glucose 73 mg/dL. A peripheral blood smear reveals eosinophilia. On urinalysis she has 2+ proteinuria but no blood, glucose, or ketones. Which of the following is the most likely diagnosis?

- A. Post-streptococcal glomerulonephritis
- B. Drug-induced interstitial nephritis
- C. IgA nephropathy
- D. Acute tubular necrosis
- E. Acute serum sickness

18-Adult polycystic kidney is:

- a-Fatal in children
- b-Autosomal dominant
- c- causing Atrophic kidney
- d-all of the above

19- Clear cell carcinoma of the kidney is associated with:

- a-Chromosome 3 abnormality
- b- Chromosome 7 abnormality
- c- Early childhood
- d- all of the above

20-Chronic renal failure occur as a complication in all of the following (except):

- a-Acute tubular necrosis
- b-Chronic GN
- c- Chronic pyelonephritis
- d- Renal stones

21-Hydronephrosis is a result of :

- A-Sudden complete obstruction
- b-Intermittent obstruction
- c- Generalized edema
- d- All of the above

22-Focal segmental glomerulosclerosis is characterized by:

- a-Loss of foot processes
- b-Nephrotic syndrome
- c- Associated with Heroin addiction
- d- all of the above

23- The following disease usually ends by Acute renal failure:

- A-Lipoid nephropathy
- b-Crescentic GN
- c- Membranous GN
- d- Post strept GN